Refeeding syndrome in a 12-year-old girl with an eating disorder and distal renal tubular acidosis

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Abstract

We experienced refeeding syndrome (RFS) with an eating disorder and distal renal tubular acidosis (dRTA). A low body mass is high-risk factors for RFS development. Therefore, frequently measurement of serum phosphorus levels at the initiation of nutrition and rapid administration of phosphate preparations are required to prevent RFS-related organ failure.

Key Clinical Message

Eating disorders and distal renal tubular acidosis are risk of refeeding sydrome. Frequent measurement of serum phosphorus levels at the initiation of nutrition and rapid administration of phosphate preparations are required to prevent organ failure.

Introduction

Refeeding syndrome (RFS) is caused by electrolyte and fluid movement upon resuming nutrition either orally, enterally, or parenterally, in severely undernourished patients.[1] Distal renal tubular acidosis (dRTA) is caused by decreased acid excretion from the distal tubules despite metabolic acidosis and a normal or mildly reduced glomerular filtration rate. We encountered a case of RFS in a patient with an underlying eating disorder and dRTA.

Case History

A 12-year-old girl visited the emergency department after vomiting for 3 days and experiencing generalized muscle weakness owing to vomiting. Her father had ulcerative colitis. She had neither growth retardation nor previous episodes of muscle weakness, although she had one previous episode of frequent vomiting. However, she had restricted her diet due to bullying at school 6 months earlier and had observed a 3.4 kg weight loss. As her serum potassium level was 1.3 mEq/L, she was hospitalized with an initial diagnosis of hypokalemic quadriplegia. Her blood pressure was 125/88 mmHg without diminished cutaneous turgor. Her body mass index (BMI) was 13.5 kg/m^2 (height, 140.3 cm; weight, 26.7 kg). Blood gas analysis upon admission revealed metabolic acidosis at pH 7.181, with a base excess of -16.3 mmol/L and a normal anion gap (14.7 mEq/L). After admission, treatment was initiated with potassium infusion of up to 0.2 mEq/kg/h and total calorie intake of 300 kcal/day by parenteral nutrition. The patient's vomiting and generalized muscle weakness resolved and there was improvement in hypokalemia and metabolic acidosis. However, on Day 3, her serum phosphorus and creatine kinase (CK) levels decreased to 1.4 mg/dL and increased to 7726 U/L, respectively (Fig. 1). Therefore, we diagnosed RFS with rhabdomyolysis. To treat RFS, oral phosphate administration (900 mg/day of dibasic sodium phosphate anhydrous) was initiated. The serum phosphorus levels normalized on Day 6. Serum CK peaked on Day 4 (12,439 U/L) but normalized on Day 7. Potassium correction continued until Day 7, with oral phosphate preparation continued for an additional 3 days. She

was discharged after 31 days in hospital with no observed organ failure. In this case, the patient's metabolic acidosis had a normal anion gap, her urinary beta 2-microglobulin level was high (64,567 μg/L), and her blood pH was lower than 7.30. The patient's urine pH did not fall below 5.5. Moreover, we found that her HCO3- excretion fraction with sodium bicarbonate load was 2.16% (normal: <3%). Her condition was complicated with dRTA. At the last follow up, neither nephrocalcinosis nor sensorineural hearing loss had been observed. Furthermore, her anti-SS-A antibody titer increased to 65.7 U/mL, which was considered to be dRTA caused by Sjogren's syndrome, rather than primary dRTA. She has continued oral administration of sodium bicarbonate and potassium preparations. The patient and her patients agreed to the publication of this case report.

Discussion

In general, when sugar is rapidly supplied to an undernourished patient, insulin is simultaneously released with phosphate, potassium, and magnesium which are taken into the cell, resulting in hypophosphatemia.[1] As a result, 2,3-diphosphoglycerate levels in erythrocytes are reduced and the oxygen affinity of hemoglobin is lowered, leading to reduced oxygen supply to peripheral tissues.[1] RFS reportedly occurs in 10-40% of undernourished patients.[2] Complications associated with RFS include heart, respiratory and/or renal failures, and evidence of skeletal muscle, nervous system, endocrine, and/or blood disorders.[3]

In this case, hypokalemia was observed due to an underlying eating disorder beginning from 6 months prior to admission, and latent dRTA. Hypokalemia and low BMI have been previously reported as high-risk factors for RFS development.[3] To prevent RFS, healthcare professionals must recognize the possibility of a patient to develop RFS and understand the risk factors from the introduction of nutrition therapy. Patients must be monitored frequently, and in case of low serum phosphorus levels, phosphoric acid preparations must be administered promptly.[4]

At the beginning of treatment, oral intake was difficult for our patient due to nausea; therefore, parenteral nutrition was initiated with a total caloric value of 300 kcal/day (approximately 11.5 kcal/kg/day). This was in line with the recommended dose for patients at high-risk for RFS at the start of nutrition, which is 5-15 kcal/kg/day.[5] The patient's calorie intake was carefully increased, but RFS developed, nonetheless. However, no severe organ failure related to RFS was observed due to the early recognition of hypophosphatemia and the prompt initiation of orally administered phosphate preparations.

In conclusion, to the best of our knowledge, this is the first reported case of a patient with RFS complicated by dRTA. Patients with a low BMI may be more likely to develop RFS when there is an underlying disease, such as dRTA. Therefore, frequent measurement of serum phosphorus levels at the initiation of nutrition and rapid administration of phosphate preparations are required to prevent RFS-related organ failure.

Author Contributions:

YS wrote the manuscript. YA performed in-hospital care for this case. HK critically reviewed the manuscript. All authors read and approved the final manuscript.

Conflicts of interest

None declared

Abstract

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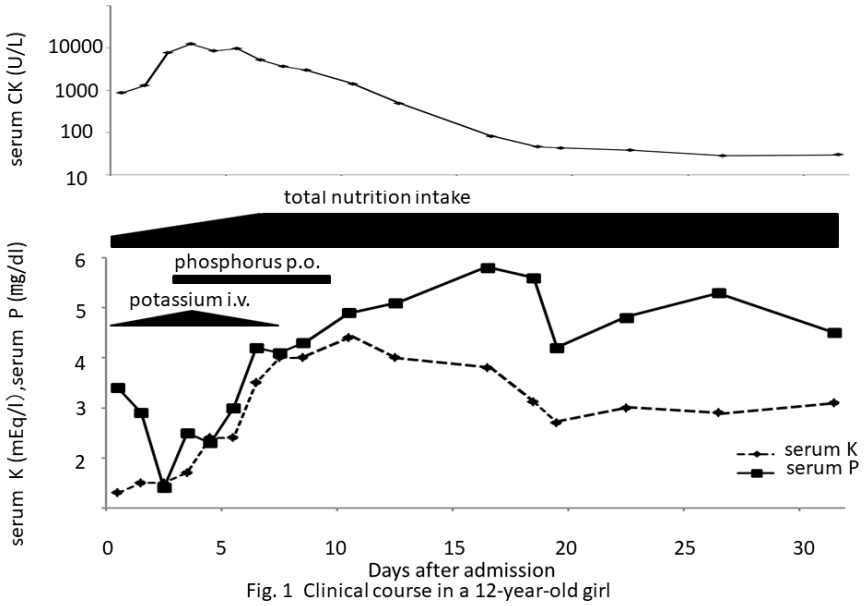
Figure Legend

Fig. 1 Clinical course in a 12-year-old girl

serum K - serum potassium, serum P - serum phosphorus, CIV continuous intravenous infusion



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CK - creatine kinase, K - potassium , serum P - phosphorus, p.o. -per os, i.v. - intravenous injection